Computer Analysis of Carotid and Brachial Pulse Waves

Effects of Age in Normal Subjects*

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IT HAS been known for some time that aging is associated with a reduction in the distensibility of the aorta and large arteries.[1,2] Recent evidence suggests that these age-related changes may be detected in man by means of the externally recorded carotid pulse wave.[3-5] Furthermore, pharmacologic agents that alter arterial distensibility change the configuration of the carotid wave in the expected direction; that is, during drug-induced reduction in distensibility the wave changes resemble those found with aging, whereas after acute increase in distensibility the reverse occurs.[5,6]

In addition to providing a possible index of arterial aging, external pulse wave recordings are of interest for the following reasons: (1) they can be recorded by a trained technician using painless, bloodless methods; (2) improvement in transducers has facilitated the obtaining of good quality tracings with rapidity and ease[7]; (3) magnetic tape recordings followed by computer analysis permit the processing of a large volume of recordings, an important consideration in population surveys or in large clinics.

In the present investigation computer technics were utilized in the analysis of the recordings. The main purpose of the study was to assess the effects of aging on the carotid and brachial pulse waves of "normal" subjects. The brachial pulse was included because it has not been studied previously with respect to aging and is less subject to artifacts produced by respiratory and other movement or by venous pulsations than the carotid pulse.

METHODS

EQUIPMENT AND PROCEDURE

Pulse wave recordings were made with a strain gauge transducer[7] consisting of a water-filled chamber surfaced on one side by a thin plastic membrane and on the opposite side by a metal diaphragm. The transducer was applied over the artery by means of a clamp adjusted to provide a loading pressure of 20 to 30 mm. Hg. The frequency response of the transducer was flat (± 5 per cent) to 20 c.p.s. Linearity and hysteresis were within ± 1 per cent over the working range of the instrument.[7]

The transducer was connected to a Sanborn carrier-wave preamplifier and recordings made on magnetic tape. Three F.M. channels of information were recorded simultaneously, the carotid and brachial pulse waves and lead I of the electrocardiogram.

Blood pressure was taken by the auscultatory method at the beginning and end of the recording session. The subjects rested supine in an air-conditioned room (74 to 78° F.) during the procedure. They were instructed to hold their breath in expiration for approximately 5 seconds while the recordings were being taken.

SUBJECTS

Carotid and brachial pulse waves were recorded in 228 male subjects classified as normal without clinically evident atherosclerotic complications, heart disease, hypertension, or diabetes mellitus. They ranged in age from 15 to 90 years and had no evidence of anemia, recent weight loss, or fever. Approximately a third were hospital personnel or students; the remainder were patients admitted for elective surgical procedures or convalescing from acute illnesses.

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METHOD OF ANALYSIS

Conversion of the analog data to digital form was carried out *by methods* previously described.[8] Computer plots were obtained from the digital tape of amplitudes against the times of the simultaneously recorded electrocardiogram and of the carotid and brachial pulse waves. The points used in the analysis were then identified on the waves by inspection as follows: the beginning of the QRS complex (Q) in the electrocardiogram, the beginning (F_1) and end (F_2) of each pulse wave, the incisura (I) and the dicrotic wave (D) (Fig. 1). F_1 and F_2 were identified as the intersection of a straight line fitted to the steepest portion of the systolic upstroke of the pulse wave with a horizontal base line drawn through the points of minimal amplitude (foot points). The point selected for the incisura was the minimum of that inflection, and for the dicrotic wave the midpoint of the succeeding positive wave. In the brachial wave only one systolic peak (P) was identified, but in the carotid wave two systolic maxima (P_1 and P_2) were identified.

TABLE I

Correlation Coefficients of Measurement Values Versus Age

Brachial Artery		Carotid Artery	
Measurement	Correlation Coefficient	Measurement	Correlation Coefficient
I/D	0.65	P_2/P_1	0.60
I-D %	−0.59	I/P_1	0.51
F_1-P %	0.46	I-D %	−0.50
I/P	0.35	I/D	0.38
Q-F_1	−0.32	F_1-P_2 %	−0.29
F_1-F_2	−0.03	F_1-P_1 %	0.10
F_1-I %	0.02	Q-F_1	−0.08
		F_1-I %	−0.03
		F_1-F_2	−0.01

F_1-F_2 = time interval between the beginning and end of the pulse wave cycle; F_1-I % = time interval from beginning of pulse wave to incisura as a percentage of total cycle length; F_1-P % = foot to peak time as a per cent of total cycle length in the brachial pulse wave; I/D = amplitude ratio of the incisura relative to the midpoint of the dicrotic wave; I-D % = time interval as a per cent of the total cycle length from the incisura to the midpoint of the dicrotic wave; I/P = amplitude ratio of incisura relative to peak in the brachial pulse wave; P_2/P_1 = amplitude ratio of the second systolic maximum relative to the first in the carotid pulse wave; Q-F_1 = interval between beginning of QRS complex in electrocardiogram and beginning of pulse wave cycle.

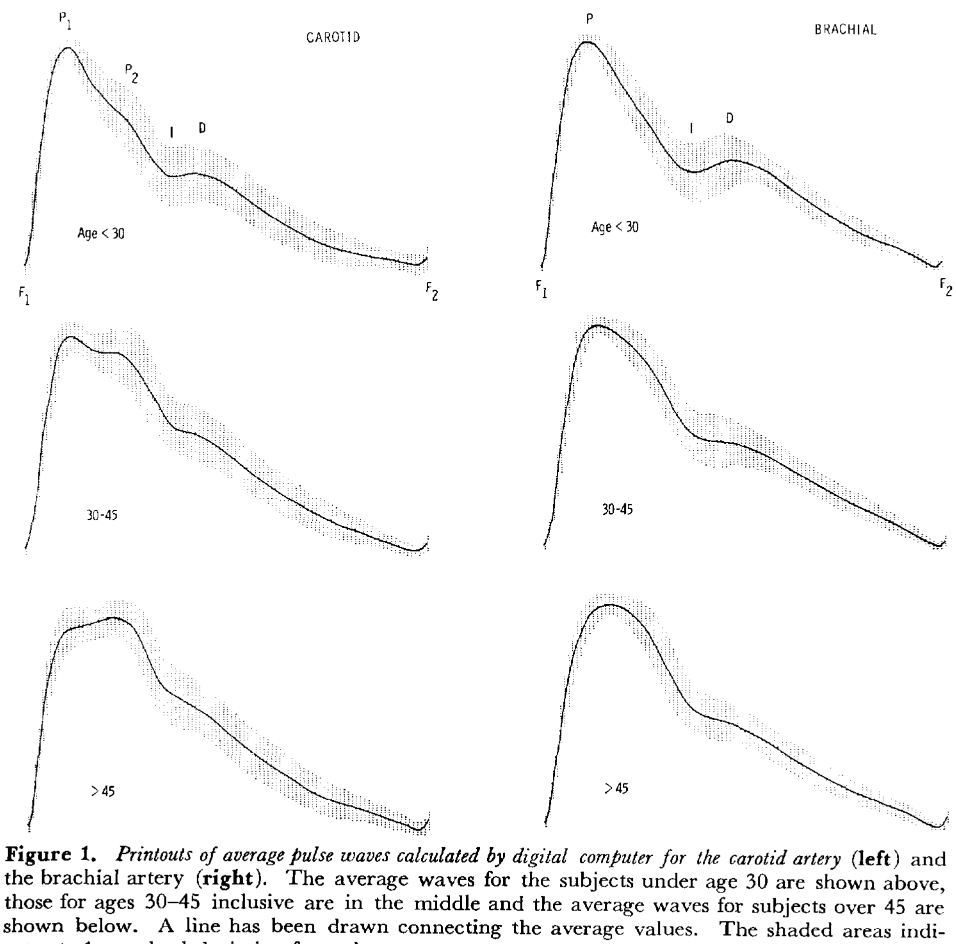

Figure 1. *Printouts of average pulse waves calculated by digital computer for the carotid artery* (**left**) and the brachial artery (**right**). The average waves for the subjects under age 30 are shown above, those for ages 30–45 inclusive are in the middle and the average waves for subjects over 45 are shown below. A line has been drawn connecting the average values. The shaded areas indicate ± 1 standard deviation from the mean.

The times of occurrence of these identified points were punched on cards. The latter plus the digital tape comprised the data input of the computer programs. From these data the various time and amplitude measurements indicated in Table I were calculated. Amplitude ratios were indicated by fractions, such as I/D, the amplitude ratio of the incisura relative to the midpoint of the dicrotic wave. Absolute time intervals were indicated by a hyphen separating the points of measurement, such as F_1-F_2, the time interval between the beginning and end of the pulse wave cycle. Relative time intervals were indicated by a hyphen and per cent sign, such as I-D %, the time interval as a per cent of the total cycle length from the incisura to the midpoint of the dicrotic wave.

To illustrate the pulse wave changes which occur with aging, average curves were constructed from the carotid and brachial waves for each of three subgroups: subjects under 30, subjects from 30 to 45 and subjects more than 45 years of age. Utilizing the computer, average curves were obtained by normalizing the amplitudes of the pulse cycles with respect to the base line and then dividing each cycle into 100 intervals. The mean and standard deviation of the amplitudes at each division point were then computed and plotted (Fig. 1).

The correlation coefficients between the various measurement values and age were calculated. Scatter diagrams were also plotted (Fig. 2 and 3).

Results

The reconstructed average pulse waves in the normal subjects revealed characteristic changes in shape with advancing age (Fig. 1). In the carotid waves the most prominent change was in the amplitude of the second systolic maximum relative to the first (P_2/P_1). In the subjects under age 30 the height of the first was of greater amplitude than the second, whereas with increasing age, the second maximum rose relative to the first. The height of the incisura relative to the first systolic maximum (I/P_1) also rose with increasing age. In the averaged brachial pulse waves the prominent changes were a decrease in amplitude of the dicrotic wave relative to the incisura (I/D) and a shortening of I-D with increasing age (Fig. 1).

The correlation coefficients calculated between the various measurement values and age are presented in Table I. The most significant changes in the brachial recordings were indicated by the correlation coefficient of 0.65 between I/D and age (Table I, Fig. 2) and of −0.59 between I-D % and age. The foot to peak time (F_1-P %) in the brachial pulse wave also increased with age, the correlation coefficient being 0.46.

In the carotid pulse wave the value for the correlation coefficient between P_2/P_1 and age was 0.60 (Table I, Fig. 2) and 0.51 between I/P_1 and age. The I-D % interval decreased with age, the correlation coefficient being −0.50.

No significant correlations were found between age and cycle length (F_1-F_2) or in ejection period as a percentage of cycle length (F_1-I %) in either the brachial or the carotid pulse waves.

Multiple correlation coefficients were determined by using measurement values from both the carotid and brachial pulse waves. The multiple correlation coefficient for carotid P_2/P_1 and brachial I/D with age was 0.71. Additional multiple correlation coefficients with age were as follows: brachial I/D and carotid I/P_1 ($r = 0.68$), brachial I-D % and carotid P_2/P_1 ($r = 0.66$) and brachial I-D % and carotid I/P_1 ($r = 0.62$).

Discussion

Carotid Pulse Wave: The principal age-related changes in the carotid pulse waves of male subjects were an increase in the amplitude of both the incisura and the second systolic maximum relative to the first systolic maximum. These carotid pulse wave changes with increasing age have been noted previously by Dontas et al.[3] and Friart.[4] Although the mechanism of these changes has not been completely elucidated, they probably reflect at least in part the decrease in distensibility of the central arterial system which occurs with aging.[5,6]

Brachial Pulse Wave: In addition to these alterations in the carotid wave the present study demonstrated that the brachial pulse also exhibits characteristic changes with age. The brachial wave is technically somewhat easier to record and is less subject to respiratory or other motion artifacts than is the carotid wave. These technical considerations may be of some importance in large scale surveys in which it is often necessary to minimize the time spent on individual recordings.

The most significant age-related changes in the brachial arterial pulse were a decrease in the amplitude and duration of the dicrotic wave. Lax and Feinberg et al.[9,10] noted a similar diminution in the dicrotic wave of the digital pulse in subjects with atherosclerosis and adults with diabetes as well as in children with diabetes mellitus. Strandness and Bell.[11]

Figure 2. *Scatter diagram* showing the relation between the amplitude ratio of the second to the first systolic maximum of the carotid pulse wave and age.

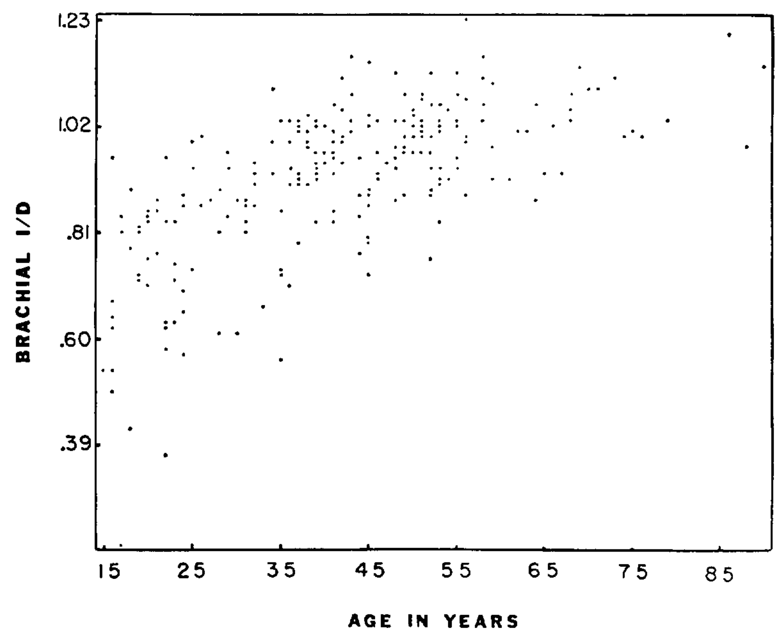

Figure 3. *Scatter diagram* showing the relation between the amplitude ratio of the incisura to the dicrotic wave of the brachial arterial pulse and age.

using a mercury resistance gauge plethysmograph, also observed diminished to absent dicrotic waves in the presence of clinically apparent atherosclerosis. Thus, alterations in the dicrotic wave of the peripheral pulse may prove to be a useful index of arterial changes associated with aging and vascular disease.

Clinical Implications: The present study indicates that the shape of the arterial pulse wave changes progressively with age from the third decade onward. Does the young individual with a pulse wave configuration "older" than his chronologic age already have the beginnings of vascular disease? The observations of Feinberg and Lax[10] in children with diabetes suggest that this may be the case. Long-term prospective studies will be needed to answer this important question. It is hoped that as a result of the present study and others made earlier, pulse wave recordings will be incorporated in longitudinal surveys currently being carried out on large population groups.

Summary

Externally recorded carotid and brachial arterial pulse waves were analyzed with the aid of a digital computer in a series of 228 male subjects free of clinically detectable cardiovascular disease. Alterations in pulse wave contours were found to be significantly correlated with age. In the carotid wave the predominant changes with age involved the relative amplitudes of the incisura and the two systolic maxima; in the brachial pulse the principal alteration with age was a diminution in the amplitude and duration of the dicrotic wave. The degree of correlation of pulse wave changes with age was approximately the same in the brachial and carotid tracings.

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